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DATE: Saturday, March 06, 2004

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☐	L3	(zinc finger near2 (regulat$3 or induction or induce or repress$3)) same (gene near2 (identif$ or detect$))	12
☐	L2	(zinc finger near2 express$3) same (gene near2 (identif$ or detect$))	13
☐	L1	zinc finger same (gene near2 identif$ or detect$)	514

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NEWS 3	SEP 09	CA/CAplus records now contain indexing from 1907 to the present
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NEWS 17	DEC 22	Additional INPI reactions and pre-1907 documents added to CAS databases
NEWS 18	DEC 22	IFIPAT/IFIUDB/IFICDB reloaded with new data and search fields
NEWS 19	DEC 22	ABI-INFORM now available on STN
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NEWS 21	JAN 27	A new search aid, the Company Name Thesaurus, available in CA/CAplus
NEWS 22	FEB 05	German (DE) application and patent publication number format changes
NEWS 23	MAR 03	MEDLINE and LMEDLINE reloaded
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FILE 'MEDLINE' ENTERED AT 12:32:29 ON 06 MAR 2004

FILE 'BIOSIS' ENTERED AT 12:32:29 ON 06 MAR 2004

=> s zinc finger and (gene (2A) (identif? or detect?)
UNMATCHED LEFT PARENTHESIS 'AND (GENE'
The number of right parentheses in a query must be equal to the
number of left parentheses.

=> s zinc finger and (gene (2A) (identif? or detect?))
L1 953 ZINC FINGER AND (GENE (2A) (IDENTIF? OR DETECT?))

=> s l1 and py<2000
1 FILES SEARCHED...
L2 530 L1 AND PY<2000

=> d 1-10 bib ab

L2 ANSWER 1 OF 530 MEDLINE on STN
AN 2000092463 MEDLINE
DN PubMed ID: 10628841
TI Yeast genes GIS1-4: multicopy suppressors of the Gal- phenotype of snf1 mig1 srb8/10/11 cells.
AU Balciunas D; Ronne H
CS Department of Plant Biology, Swedish University of Agricultural Sciences, Uppsala Genetic Center.
SO Molecular & general genetics : MGG, **(1999 Dec)** 262 (4-5) 589-99.
Journal code: 0125036. ISSN: 0026-8925.
CY GERMANY: Germany, Federal Republic of
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200001
ED Entered STN: 20000209
Last Updated on STN: 20030206
Entered Medline: 20000131
AB Cyclin C and the cyclin C-dependent protein kinase are associated with the RNA polymerase II Mediator complex, which regulates initiation of transcription in response to signals from activators and repressors bound to upstream promoter elements. Disruption of the corresponding genes, SRB11 and SRB10, in budding yeast causes a reduction in expression of the GAL genes, which is particularly pronounced in a mig1 snf1 background. We have screened two yeast genomic libraries for genes that can suppress this phenotype when overexpressed. Seven suppressor **genes** were **identified**, GIS1-7. GIS1 encodes one of two related **zinc -finger** proteins, which also share two other highly conserved domains present in several eukaryotic transcription factors. GIS2 encodes a homologue of the mammalian CNBP and fission yeast Byr3 proteins. GIS3 and GIS4 predict proteins with no obvious similarities to any known proteins. GIS5-7 are identical to the previously described genes PDE2,

SGE1 and TUB3, respectively. None of the suppressor genes seem to be involved in Mediator function. Instead, we find that the GIS1, GIS2 and GIS4 genes interact with the CDC25 gene, indicating a possible involvement of these genes in the RAS/cAMP signaling pathway.

L2 ANSWER 2 OF 530 MEDLINE on STN
AN 2000035820 MEDLINE
DN PubMed ID: 10571043
TI An enhancer trap line identifies the Drosophila homolog of the L37a ribosomal protein.
AU Gaines P; Woodard C T; Carlson J R
CS Department of Biology, Yale University, New Haven, CT 06520-8103, USA.
SO Gene, **(1999 Oct 18)** 239 (1) 137-43.
Journal code: 7706761. ISSN: 0378-1119.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199912
ED Entered STN: 20000113
Last Updated on STN: 20000113
Entered Medline: 19991210
AB A **gene identified** from an enhancer trap screen is shown to encode the Drosophila melanogaster homolog of the L37a ribosomal protein. The predicted 92 amino-acid sequence of this protein is 78% identical to mammalian L37a proteins, and contains a conserved Cys-X2 Cys-X14-Cys-X2-Cys **zinc finger** motif that may be involved in interactions with ribosomal RNA. The Drosophila L37a homolog is a single copy gene comprised of four exons and is ubiquitously expressed throughout the animal. Cytological localization reveals that Drosophila L37a maps to position 25C1-3, very near the previously described Minute mutation M(2)25C.

L2 ANSWER 3 OF 530 MEDLINE on STN
AN 2000033544 MEDLINE
DN PubMed ID: 10564808
TI Characterization of the mouse Kid1 **gene** and **identification** of a highly related gene, Kid2.
AU Tekki-Kessaris N; Bonventre J V; Boulter C A
CS University of Cambridge, Department of Genetics, Downing Street, Cambridge, UK.
NC DK 39773 (NIDDK)
SO Gene, **(1999 Nov 15)** 240 (1) 13-22.
Journal code: 7706761. ISSN: 0378-1119.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-AF184111; GENBANK-AF184112
EM 200001
ED Entered STN: 20000124
Last Updated on STN: 20000124
Entered Medline: 20000111
AB Kid1 encodes a **zinc finger** protein that has been implicated in renal cell differentiation. Levels of Kid1 mRNA correlate with maturation of kidney tubule epithelia in rat post-natal kidney development and during kidney regeneration following injury. KID1 is a putative transcriptional repressor, containing a KRAB domain at its amino terminus that mediates transcriptional repression in transient cell transfection assays when fused to a heterologous DNA-binding domain. In this paper, we describe the isolation and characterization of the mouse homologue of Kid1 and the identification of a novel highly related mouse

gene, Kid2, Kid1 and Kid2 are tightly linked on mouse chromosome 11 and show conservation across mammals. Both genes are expressed predominantly in the mouse adult kidney and brain, but transcripts are also detected in embryonic brain, kidney, gut and lung, suggesting an additional role for these genes during mouse development.

L2 ANSWER 4 OF 530 MEDLINE on STN
AN 2000028757 MEDLINE
DN PubMed ID: 10563017
TI **Detecting** and characterizing **gene** conversions between multigene family members.
AU Drouin G; Prat F; Ell M; Clarke G D
CS Departement de biologie, Universite d'Ottawa, Ontario, Canada.. guy@bio01.bio.uottawa.ca
SO Molecular biology and evolution, **(1999 Oct)** 16 (10) 1369-90.
Journal code: 8501455. ISSN: 0737-4038.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199912
ED Entered STN: 20000113
Last Updated on STN: 20000113
Entered Medline: 19991208
AB We used a variety of methods to **detect** known **gene** conversions in the actin gene families of five angiosperm species, the beta-globin gene families of two primate species, and the Zfx/Zfy gene families of seven mammalian species. Our goal was to devise a working strategy which would allow the analysis of the members of a multigene family in order to determine whether there had been gene conversions between its members, **identify** the **genes** involved in the gene conversions, establish the lengths of the converted regions, and determine the polarities of the gene conversions. We show that three phylogenetic methods and the homoplasy test of Maynard Smith and Smith perform relatively poorly on our data sets because the sequences we analyzed had large levels of multiple substitutions. The method of Sawyer, the compatibility method of Jakobsen and Easteal, the partition matrix method of Jakobsen, Wilson, and Easteal, and the co-double method of Balding, Nichols, and Hunt can be used to **identify** the **genes** which have been involved in gene conversions. The co-double method is more powerful than other methods but requires orthologous sequences from related species. Compatibility, phylogenetic, and nucleotide substitution distribution statistics methods can be used to identify the location of the converted region(s). Site-by-site compatibility analyses can also be used to identify the direction of the conversion event(s). Combinations of these methods can therefore be used to establish the presence, locations, and polarities of gene conversions between multigene family members.

L2 ANSWER 5 OF 530 MEDLINE on STN
AN 2000012750 MEDLINE
DN PubMed ID: 10544010
TI **Identification** and **gene** structure of a novel human PLZF-related transcription factor gene, TZFP.
AU Lin W; Lai C H; Tang C J; Huang C J; Tang T K
CS Institute of Biomedical Sciences, Academia Sinica, Taipei, 115, Taiwan.. wenlin@ibms.sinica.edu.tw
SO Biochemical and biophysical research communications, **(1999 Nov 2)** 264 (3) 789-95.
Journal code: 0372516. ISSN: 0006-291X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)

LA English
FS Priority Journals
OS GENBANK-AF130255
EM 199912
ED Entered STN: 20000113
Last Updated on STN: 20000113
Entered Medline: 19991222
AB A novel cDNA clone was identified through yeast two-hybrid experiments. Following cross-examination between the cDNA clones, EST clones, and the cosmid clone, we could digitally assemble a new **zinc finger** transcription factor gene. This predicted gene has a cDNA size of about 1960 bp and is translated into a 487-amino-acid protein. According to database analysis, this gene contains three C2H2 **zinc finger** motifs and is highly related to human PLZF (promyelocytic leukemia **zinc finger** protein). The full-length coding region of the gene was isolated, and its sequences were confirmed by DNA sequencing. Interestingly, one splicing variant lacking exon III was also identified. Northern blot analysis revealed that this gene is mainly expressed in human testis. In conclusion, we have identified a new member of the PLZF **zinc finger** protein family, the testis **zinc finger** protein (TZFP), which is mainly expressed in testis tissue.

L2 ANSWER 6 OF 530 MEDLINE on STN
AN 2000011288 MEDLINE
DN PubMed ID: 10542327
TI Isolation, cloning, and expression of a new murine **zinc finger** encoding gene.
AU Prost J F; Negre D; Cornet-Javaux F; Cortay J C; Cozzone A J; Herbage D; Mallein-Gerin F
CS Institut de Biologie et Chimie des Proteines, CNRS UPR 412, 7 passage du Vercors 69367, Lyon, France.
SO Biochimica et biophysica acta, **(1999 Oct 28)** 1447 (2-3) 278-83.
Journal code: 0217513. ISSN: 0006-3002.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-AF149093
EM 199912
ED Entered STN: 20000113
Last Updated on STN: 20000113
Entered Medline: 19991208
AB With the aim of **identifying genes** involved in cartilage differentiation, we have used a subtractive hybridization strategy with cDNAs from a chondrocytic cell line (MC615) and mRNAs from a mesenchymal precursor cell line (10T1/2). We have isolated a cDNA clone representing a novel mouse gene. The predicted 368-amino acid protein, designated ZF-12, contains four C(2)H(2)-type **zinc finger** motifs and one region homologous to the LeR domain, a finger-associated structural domain. ZF-12 mRNAs are expressed during embryonic development and in different organs in adult, including rib cartilage. These data suggest that ZF-12 might play an important role not only in cartilage differentiation, but also in basic cellular processes.

L2 ANSWER 7 OF 530 MEDLINE on STN
AN 1999452777 MEDLINE
DN PubMed ID: 10523314
TI The Saccharomyces cerevisiae MER3 gene, encoding a novel helicase-like protein, is required for crossover control in meiosis.
AU Nakagawa T; Ogawa H

CS Department of Biology, Graduate School of Science, Osaka University, Toyonaka, Osaka 560-0043, Japan.
SO EMBO journal, **(1999 Oct 15)** 18 (20) 5714-23.
Journal code: 8208664. ISSN: 0261-4189.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199912
ED Entered STN: 20000113
Last Updated on STN: 20000113
Entered Medline: 19991206
AB The MER3 **gene** is **identified** as a novel meiosis-specific gene, whose transcript is spliced in an MRE2/MER1-dependent manner. The predicted Mer3 protein contains the seven motifs characteristic of the DExH-box type of helicases as well as a putative **zinc finger**. Double strand breaks (DSBs), the initial changes of DNA in meiotic recombination, do not disappear completely and are hyperresected late in mer3 meiosis, indicating that MER3 is required for the transition of DSBs to later intermediates. A mer3 mutation reduces crossover frequencies, and the remaining crossovers show random distribution along a chromosome, resulting in a high incidence of non-disjunction of homologous chromosomes at the first meiotic division. MER3 appears to be very important for both the DSB transition and crossover control.

L2 ANSWER 8 OF 530 MEDLINE on STN
AN 1999446483 MEDLINE
DN PubMed ID: 10518215
TI SLUG, a ces-1-related **zinc finger** transcription factor gene with antiapoptotic activity, is a downstream target of the E2A-HLF oncoprotein.
AU Inukai T; Inoue A; Kurosawa H; Goi K; Shinjyo T; Ozawa K; Mao M; Inaba T; Look A T
CS Department of Experimental Oncology, St. Jude Children's Research Hospital, Memphis, Tennessee 38105, USA.
NC CA 21765 (NCI)
CA 59571 (NCI)
SO Molecular cell, **(1999 Sep)** 4 (3) 343-52.
Journal code: 9802571. ISSN: 1097-2765.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199910
ED Entered STN: 20000111
Last Updated on STN: 20000111
Entered Medline: 19991029
AB The E2A-HLF fusion gene transforms human pro-B lymphocytes by interfering with an early step in apoptotic signaling. In a search for E2A-HLF-responsive **genes**, we **identified** a **zinc finger** transcription factor, SLUG, whose product belongs to the Snail family of developmental regulatory proteins. Importantly, SLUG bears close homology to the CES-1 protein of C. elegans, which acts downstream of CES-2 in a neuron-specific cell death pathway. Consistent with the postulated role of CES-1 as an antiapoptotic transcription factor, SLUG was nearly as active as Bcl-2 or Bcl-xL in promoting the survival of IL-3-dependent murine pro-B cells deprived of the cytokine. We conclude that SLUG is an evolutionarily conserved transcriptional repressor whose activation by E2A-HLF promotes the aberrant survival and eventual malignant transformation of mammalian pro-B cells otherwise slated for apoptotic death.

L2 ANSWER 9 OF 530 MEDLINE on STN
AN 1999431604 MEDLINE
DN PubMed ID: 10502321
TI A KRAB **zinc finger** protein gene is the potential target of 19q13 translocation in benign thyroid tumors.
AU Rippe V; Belge G; Meiboom M; Kazmierczak B; Fusco A; Bullerdiek J
CS Center for Human Genetics and Genetic Counseling, University of Bremen, Bremen, Germany.
SO Genes, chromosomes & cancer, **(1999 Nov)** 26 (3) 229-36.
Journal code: 9007329. ISSN: 1045-2257.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-AF260531
EM 200002
ED Entered STN: 20000218
Last Updated on STN: 20000218
Entered Medline: 20000210
AB In an attempt to **identify** the target **gene** of specific translocations involving chromosomal band 19q13 in benign follicular thyroid tumors, we have used two cell lines derived from benign thyroid tumors showing translocations with 19q13 breakpoints for fluorescence in situ hybridization mapping studies with cosmid and PAC clones located in a 400-kbp region. The breakpoints of the chromosome 19 abnormalities mapped within a 140-kb segment covered by a single PAC clone. Sequencing of part of this PAC clone allowed us to establish the cDNA sequence and the genomic structure of a candidate gene located in close vicinity to the breakpoints. The gene that we tentatively refer to as RITA (rearranged in thyroid adenomas) belongs to the KRAB **zinc finger** protein coding genes. From our results we have concluded that in the two cell lines investigated the breaks have occurred either within the 5' untranslated region of RITA or in its close 5' vicinity. By Northern blot analyses two transcripts of about 4.7 kbp and 5 kbp were detected in normal thyroid tissue as well as in other normal tissues tested. An additional 2.1-kbp transcript was found only in testicular tissue. In contrast to all normal tissues, both cell lines with 19q aberrations expressed larger transcripts of approximately 5.5 kbp and 6.2 kbp. From the close vicinity to the breakpoint region, the expression patterns of the gene, and its type, we consider RITA a strong candidate target gene of the specific 19q aberrations in benign thyroid tumors.

L2 ANSWER 10 OF 530 MEDLINE on STN
AN 1999409666 MEDLINE
DN PubMed ID: 10481940
TI Differentially expressed genes associated with the metastatic phenotype in breast cancer.
AU Kirschmann D A; Seftor E A; Nieva D R; Mariano E A; Hendrix M J
CS Department of Anatomy and Cell Biology, Iowa Cancer Center, College of Medicine, The University of Iowa, Iowa City 52242-1109, USA.
NC CA59702 (NCI)
SO Breast cancer research and treatment, **(1999 May)** 55 (2) 127-36.
Journal code: 8111104. ISSN: 0167-6806.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199910
ED Entered STN: 19991101
Last Updated on STN: 19991101

Entered Medline: 19991021

AB We have previously shown that human breast carcinoma cells demonstrating an interconverted phenotype, where keratin (epithelial marker) and vimentin (mesenchymal marker) intermediate filaments are both expressed, have an increased ability to invade a basement membrane matrix in vitro. This increase in invasive potential has been demonstrated in MDA-MB-231 cells, which constitutively express keratins and vimentin, and in MCF-7 cells transfected with the mouse vimentin gene (MoVi). However, vimentin expression alone is not sufficient to confer the complete metastatic phenotype in MoVi cells, as determined by orthotopic administration. Thus, in the present study, differential display analysis was utilized to **identify genes** that are associated with the invasive and/or metastatic phenotype of several human breast cancer cell lines. Forty-four of 84 PCR fragments were differentially expressed as assessed by Northern hybridization analysis of RNA isolated from MCF-7, MoVi, and MB-231 cell lines. Polyadenylated RNA from a panel of poorly invasive, invasive/non-metastatic, and invasive/metastatic breast carcinoma cell lines was used to differentiate between cell-specific gene expression and genes associated with the invasive and/or metastatic phenotype(s). We observed that lysyl oxidase and a **zinc finger** transcription factor were expressed only in the invasive and/or metastatic cell lines; whereas, a thiol-specific antioxidant and a heterochromatin protein were down-regulated in these cells. In contrast, tissue factor was expressed only in breast carcinoma cell lines having the highest invasive potential. These results suggest that specific genes involved in breast cancer invasion and metastasis can be separated by differential display methodology to elucidate the molecular basis of tumor cell progression.

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=> d his

     (FILE 'HOME' ENTERED AT 12:32:19 ON 06 MAR 2004)

     FILE 'MEDLINE, BIOSIS' ENTERED AT 12:32:29 ON 06 MAR 2004
L1           953 S ZINC FINGER AND (GENE (2A) (IDENTIF? OR DETECT?))
L2           530 S L1 AND PY<2000

=> s (zinc finger (2a) express?) and (gene (2a) (identif? or detect?))
L3           56 (ZINC FINGER (2A) EXPRESS?) AND (GENE (2A) (IDENTIF? OR DETECT?)
                )

=> s l3 and py<2000
L4           24 L3 AND PY<2000

=> duplicate remove l4
DUPLICATE PREFERENCE IS 'MEDLINE, BIOSIS'
KEEP DUPLICATES FROM MORE THAN ONE FILE?  Y/(N):n
PROCESSING COMPLETED FOR L4
L5            15 DUPLICATE REMOVE L4 (9 DUPLICATES REMOVED)

=> d 1-15 bib ab

L5   ANSWER 1 OF 15  BIOSIS  COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN   2000:75659  BIOSIS
DN   PREV200000075659
TI   Molecular cloning of six novel Kruppel-like zinc finger genes from
     hematopoietic cells and identification of a novel transregulatory domain
     KRNB.
AU   Han, Ze-Guang; Zhang, Qing-Hua; Ye, Min; Kan, Li-Xin; Gu, Bai-Wei; He,
     Kai-Li; Shi, Shao-Lin; Zhou, Jun; Fu, Gang; Mao, Mao; Chen, Sai-Juan; Yu,
     Long; Chen, Zhu [Reprint author]
```

CS Shanghai Inst. of Hematology, Rui-Jin Hospital, Shanghai Second Medical University, 197 Rui Jin Road II, Shanghai, 200025, China
SO Journal of Biological Chemistry, (Dec. 10, 1999) Vol. 274, No. 50, pp. 35741-35748. print.
CODEN: JBCHA3. ISSN: 0021-9258.
DT Article
LA English
OS Genbank-AF038951; Genbank-AF038964; Genbank-AF054180; Genbank-AF067164; Genbank-AF067165; Genbank-AF070651
ED Entered STN: 23 Feb 2000
Last Updated on STN: 3 Jan 2002
AB To clone **zinc finger** genes **expressed** in hematopoietic system, we designed primers based on conserved Cys2/His2 zinc finger sequences to amplify corresponding domains from mRNA of normal bone marrow and leukemia cell line NB4. DNA fragments of novel zinc finger genes were chosen and used as probe pool to screen cDNA libraries or subject to rapid amplification of cDNA ends in order to obtain full-length cDNA. Six cDNAs including whole open reading frame of zinc finger proteins, named as ZNF191, ZNF253 (BMZF-1), ZNF255 (BMZF-2), ZNF256 (BMZF-3), ZNF257 (BMZF-4), and ZNF254 (BMZF-5) were obtained. All six belong to the Kruppel-like zinc finger gene family, and typical transcriptional regulatory motifs exist in the N-terminal moiety, such as the SCAN box in ZNF191, and the KRAB domains in ZNF253, ZNF254, ZNF256, and ZNF257. A previously undefined sequence nominated as Kruppel-related novel box, which may represent a new transregulatory motif, was revealed at the N terminus of ZNF255. The transregulatory function of non-zinc finger regions of ZNF191, ZNF253, and ZNF255 were addressed in yeast and mammalian cells. The results indicated that ZNF255 might be a conditional transactivator, whereas ZNF253 and ZNF191 displayed a suppressive effect on the transcription in yeast and/or mammalian systems.

L5 ANSWER 2 OF 15 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 1998:428140 BIOSIS
DN PREV199800428140
TI Identification and mutation analysis of a cochlear-**expressed, zinc finger** protein gene at the DFNB7/11 and dn hearing-loss-loci on human chromosome 9q and mouse chromosome 19.
AU Scott, D. A.; Greinwald, J. H., Jr.; Marietta, J. R.; Drury, S.; Swiderski, R. E.; Vinas, A.; Deangelis, M. M.; Carmi, R.; Ramesh, A.; Kraft, M. L.; Elbedour, K.; Skworak, A. B.; Friedman, R. A.;, Srisailapathy, C. R., Srikumari; Verhoeven, K.; Van Camp, G.; Lovett, M.; Deininger, P. L.; Batzer, M. A.; Morton, C. C.; Keats, B. J.; Smith, R. J. H. [Reprint author]; Sheffield, V. C.
CS Univ. Iowa Hosp. Clinics, 200 Hawkins Drive, Iowa City, IA 52242-1078, USA
SO Gene (Amsterdam), (July 30, 1998) Vol. 215, No. 2, pp. 461-469. print.
CODEN: GENED6. ISSN: 0378-1119.
DT Article
LA English
OS Genbank-AF062346; Genbank-AF062347
ED Entered STN: 7 Oct 1998
Last Updated on STN: 7 Oct 1998
AB The DFNB7/11 locus for autosomal recessive non-syndromic hearing loss (ARNSHL) has been mapped to an approx. 1.5 Mb interval on human chromosome 9q13-q21. We have determined the cDNA sequence and genomic structure of a novel cochlear-expressed gene, ZNF216, that maps to the DFNB7/11 interval. The mouse orthologue of this gene maps to the murine dn (deafness) locus on mouse chromosome 19. The ZNF216 gene is highly conserved between human and mouse, and contains two regions that show homology to the putative zinc finger domains of other proteins. To determine if mutations in ZNF216 might be the cause of hearing loss at the DFNB7/11 locus, we screened the coding region of this gene in DFNB7/11 families by direct sequencing. No potential disease-causing mutations were found. In

addition, Northern blot analysis showed no difference in ZNF216 transcript size or abundance between dn and control mice. These data suggest that the ZNF216 gene is unlikely to be responsible for hearing loss at the DFNB7/11 and dn loci.

L5 ANSWER 3 OF 15 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 1999:35530 BIOSIS
DN PREV199900035530
TI Azf1p is a nuclear-localized zinc-finger protein that is preferentially expressed under non-fermentative growth conditions in Saccharomyces cerevisiae.
AU Stein, Torsten; Kricke, Joern; Becher, Dietmar; Lisowsky, Thomas [Reprint author]
CS Botanisches Inst. I, Heinrich-Heine-Univ. Duesseldorf, Universitaetsstrasse 1, D-40225 Duesseldorf, Germany
SO Current Genetics, (Oct., 1998) Vol. 34, No. 4, pp. 287-296. print. CODEN: CUGED5. ISSN: 0172-8083.
DT Article
LA English
ED Entered STN: 3 Feb 1999
Last Updated on STN: 3 Feb 1999
AB In previous studies the AZF1 **gene** has been **identified** as a second high-copy number suppressor for a special mutant of the gene for the mitochondrial core enzyme of RNA polymerase. The first high-copy number suppressor of this mutant turned out to be the specificity factor MTF1 for mitochondrial transcription. Up to now, the influence of AZF1 on mitochondrial transcription, its precise localization in the cell and the regulation of its expression has not been determined. The putative protein contains a long stretch of poly-asparagine amino acids and a typical zinc-finger domain for DNA binding. These characteristic structural features were used to create the abbreviation AZF1 (A sparagine-rich Zinc Finger protein). An initial computer analysis of the sequence gave no conclusive results for the presence of a mitochondrial import sequence or a typical nuclear-targeting sequence. A recent more-detailed analysis identified a possible nuclear localization signal in the middle of the protein. Disruption of the gene shows no effect on plates with glucose-rich medium or glycerol. In this report a specific polyclonal antibody against AZF1 p was prepared and used in cell-fractionation experiments and in electron-microscopic studies. Both of these clearly demonstrate that the AZFI protein is localized exclusively in the nucleus of the yeast cell. Northern analysis for the expression of the AZF1 messenger RNA under different growth conditions was therefore performed to obtain new insights into the regulation of this gene. Together with the respective protein-expression analysis these data demonstrate that Azf1p is preferentially synthezised in higher amounts under non-fermentable growth conditions. Over-expression of Azf1p in the yeast cell does not influence the expression level of the mitochondrial transcription factor Mtf1p, indicating that the influence of Azf1p on the suppression of the special mitochondrial RNA polymerase mutant is an indirect one. Subcellular investigation of the deletion mutant by electron microscopy identifies specific ultrastructural cell-division defects in comparison to the wild-type.

L5 ANSWER 4 OF 15 MEDLINE on STN DUPLICATE 1
AN 1999117955 MEDLINE
DN PubMed ID: 9919311
TI Molecular cloning of a zinc finger gene eZNF from a human inner ear cDNA library, and in situ expression pattern of its mouse homologue in mouse inner ear.
AU Jacob A N; Manjunath N A; Bray-Ward P; Kandpal R P
CS Fels Institute for Cancer Research and Molecular Biology, Temple University School of Medicine, Philadelphia, Pennsylvania 19140, USA.

NC DC10682 (NIDCD)
HG00272 (NHGRI)
SO Somatic cell and molecular genetics, **(1998 Mar)** 24 (2) 121-9.
Journal code: 8403568. ISSN: 0740-7750.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-AF116030
EM 199902
ED Entered STN: 19990223
Last Updated on STN: 20000303
Entered Medline: 19990211
AB We have isolated and characterized the cDNA for eZNF, a **zinc finger** gene **expressed** in human inner ear, from a kinetically enriched human inner ear cDNA library. The sequence of full length cDNA was determined and its expression pattern characterized. A high degree of homology is shared between eZNF and rat transcription factor Kid-1. It belongs to the C2H2 class of zinc finger genes, contains a Kruppel-associated box (KRAB) domain near the N-terminus, and has consensus sites for phosphorylation. The gene is expressed in kidney and inner ear structures of mouse and human as determined by Northern blot analysis. In situ hybridization was used to demonstrate specific expression of the mouse eZNF homologue in epithelial layers of the saccule, semicircular canals, and the cochlea of newborn mice. The genomic clone corresponding to the cDNA was isolated and used for fluorescence in situ hybridization to localize it to human chromosome 5qter. The **identification** of **genes** expressed in human inner ear by representational difference analysis, their chromosomal location, and expression pattern of their homologues in developing mouse inner ear comprise a strategy that can potentially **identify genes** important in hearing and deafness.

L5 ANSWER 5 OF 15 MEDLINE on STN DUPLICATE 2
AN 1999026125 MEDLINE
DN PubMed ID: 9806829
TI Cloning and mapping of ZNF231, a novel brain-specific gene encoding neuronal double **zinc finger** protein whose **expression** is enhanced in a neurodegenerative disorder, multiple system atrophy (MSA).
AU Hashida H; Goto J; Zhao N; Takahashi N; Hirai M; Kanazawa I; Sakaki Y
CS Department of Neurology, Graduate School of Medicine, University of Tokyo, Tokyo, Japan.. hhashida-tky@umin.ac.jp
SO Genomics, **(1998 Nov 15)** 54 (1) 50-8.
Journal code: 8800135. ISSN: 0888-7543.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-AF052224
EM 199901
ED Entered STN: 19990115
Last Updated on STN: 19990115
Entered Medline: 19990107
AB A novel brain-specific gene, neuronal double zinc finger protein (ZNF231), was cloned and mapped. We used the high-density cDNA filter method to analyze the gene-expression profile in brains with multiple system atrophy (MSA). MSA is a sporadic progressive neurodegenerative disease characterized clinically by cerebellar symptoms, parkinsonism, autonomic dysfunction, or their various combinations, but its pathogenesis has yet to be clarified. In total, 8300 cDNA clones were screened, and a novel **gene**, ZNF231, was **identified**, whose expression was

elevated in cerebella of patients with MSA. Its transcript is approximately 16 kb long and encodes an open reading frame of 3926 amino acid residues that has several interesting motifs; two glycine-proline dipeptide repeats (aa 22-32 and aa 61-74), a pair of homologous C8 double zinc finger motifs (aa 169-226 and aa 465-521), a leucine zipper motif (aa 561-582), a SH3 domain-binding motif (aa 825-831), two nuclear targeting signals (aa 1011-1028 and aa 1071-1091), two glutamine-rich domains (aa 2428-2473 and aa 3775-3804), and a histidine-rich domain (aa 3597-3682). These features suggest that the new gene encodes a nuclear protein or transcription regulator. Northern blot and RT-PCR analyses showed that its expression is specific to the brain and apparently restricted to the neurons. Elevation of ZNF231 expression may be involved in the pathogenesis of multiple system atrophy. The gene for ZNF231 is located on chromosome 3p21.

L5 ANSWER 6 OF 15 MEDLINE on STN DUPLICATE 3
AN 1998355768 MEDLINE
DN PubMed ID: 9688932
TI Targeted identification of **zinc finger** genes **expressed** in rat lungs.
AU Dovat S; Gilbert K A; Petrovic-Dovat L; Rannels D E
CS Department of Pediatrics, The Pennsylvania State University College of Medicine, Hershey, Pennsylvania 17033, USA.
NC HL-08954 (NHLBI)
HL-20344 (NHLBI)
SO American journal of physiology, **(1998 Jul)** 275 (1 Pt 1) L30-7.
Journal code: 0370511. ISSN: 0002-9513.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199808
ED Entered STN: 19980903
Last Updated on STN: 19980903
Entered Medline: 19980825
AB Control of alveolar cell growth and differentiation after pneumonectomy likely involves changes in expression of regulatory genes, including those encoding zinc finger (ZF) proteins. To explore this premise, total RNA from the lungs of control and pneumonectomized rats was reverse transcribed; PCRs were performed with degenerate primers corresponding to amino acid sequences HTGEKP and CPECGK(N), which are evolutionarily conserved among ZF genes. Reaction products corresponding to three and four ZF units were isolated and cloned. Sixteen clones were sequenced and found to represent rat lung ZF genes: six clones were highly similar or identical to known ZF genes and ten clones showed lower homology to known ZF genes and thus appear to represent new members of the ZF family. Northern analysis demonstrated differential expression of some ZF genes after pneumonectomy. Thus a PCR-based strategy with primers derived from evolutionarily conserved ZF protein sequences efficiently **identifies** ZF **genes** expressed in lung, some of which may play a role in cellular growth and differentiation.

L5 ANSWER 7 OF 15 MEDLINE on STN DUPLICATE 4
AN 97223484 MEDLINE
DN PubMed ID: 9070305
TI Cloning of human and mouse cDNAs encoding novel **zinc finger** proteins **expressed** in cerebellum and hippocampus.
AU Yasojima K; Tsujimura A; Mizuno T; Shigeyoshi Y; Inazawa J; Kikuno R; Kuma K; Ohkubo K; Hosokawa Y; Ibata Y; Abe T; Miyata T; Matsubara K; Nakajima K; Hashimoto-Gotoh T
CS Department of Biochemistry and Molecular Genetics, Kyoto Prefectural

University of Medicine, Japan.
SO Biochemical and biophysical research communications, **(1997 Feb 13)** 231 (2) 481-7.
Journal code: 0372516. ISSN: 0006-291X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-D76444; GENBANK-D76445
EM 199704
ED Entered STN: 19970424
Last Updated on STN: 19980206
Entered Medline: 19970417
AB We **identified** a novel **gene**, kf-1, highly expressed in the normal cerebellum but not in the cerebral cortex, the expression of which could have been augmented in the cerebral cortex of a sporadic Alzheimer's disease patient. We cloned human and mouse entire kf-1 cDNAs encoding conserved 79 kDa proteins containing a zinc-binding RING-H2 finger motif at the carboxy-terminus as found in acetylcholine receptor-associated protein (RAPsyn). The 3'-untranslated regions are highly conserved between human and mouse as to constitute a common mRNA secondary structure. In situ hybridization analysis of mouse brain sections revealed strong kf-1 expression in the cerebellum and hippocampus. We propose that KF-1 is involved in membranous protein-sorting apparatus similarly to RAPsyn. We mapped the human kf-1 gene to 2p11.2.

L5 ANSWER 8 OF 15 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 1998:174527 BIOSIS
DN PREV199800174527
TI Salt tolerance in crop plants: New approaches through tissue culture and gene regulation.
AU Winicov, Ilga [Reprint author]; Bastola, Dhundy R.
CS Dep. Microbiology, Univ. Nevada Reno, Reno, NV 89557, USA
SO Acta Physiologiae Plantarum, (1997) Vol. 19, No. 4, pp. 435-449. print.
CODEN: APPLDE. ISSN: 0137-5881.
DT Article
General Review; (Literature Review)
LA English
ED Entered STN: 20 Apr 1998
Last Updated on STN: 20 Apr 1998
AB Recent approaches to study of salinity tolerance in crop plants have ranged from genetic mapping to molecular characterization of gene products induced by salt/drought stress. Transgenic plant design has allowed to test the effects of overexpression of specific prokaryotic or plant genes that are known to be up-regulated by salt/drought stress. This review summarizes current progress in the field in the context of adaptive metabolic and physiological responses to salt stress and their potential role in long term tolerance. Specifically considered are gene activation by salt, in view of proposed avenues for improved salt tolerance and the need to ascertain the additional influences of developmental regulation of such genes. Discussion includes the alternate genetic strategy we have pursued for improving salinity tolerance in alfalfa (Medicago saliva L.) and rice (Oryza sativa L.). This strategy combines single-step selection of salt-tolerant cells in culture, followed by regeneration of salt-tolerant plants and **identification** of **genes** important in conferring salt tolerance. We have postulated that activation or improved expression of a subset of genes encoding functions that are particularly vulnerable under conditions of salt-stress could counteract the molecular effects of such stress and could provide incremental improvements in tolerance. We have proceeded to identify the acquired specific changes in gene regulation for our salt-tolerant mutant

cells and plants. One particularly interesting and novel gene isolate from the salt-tolerant cells is Alfin1, which encodes a putative **zinc-finger** regulatory protein, **expressed** predominantly in roots. We have demonstrated that this protein binds DNA in a sequence specific manner and may be potentially important in gene regulation in roots in response to salt and an important marker for salt tolerance in crop plants.

L5 ANSWER 9 OF 15 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 1998:180007 BIOSIS
DN PREV199800180007
TI Cloning, expression and mapping of a novel RING-finger gene (RNF5), a human homologue of a putative zinc-finger gene from Caenorhabditis elegans.
AU Kyushiki, H. [Reprint author]; Kuga, Y.; Suzuki, M.; Takahashi, E.; Horie, M.
CS Otsuka GEN Res. Inst., Otsuka Pharm. Co. Ltd., 463-10 Kagasuno, Kawauchi-cho, Tokushima 771-01, Japan
SO Cytogenetics and Cell Genetics, (1997) Vol. 79, No. 1-2, pp. 114-117. print.
CODEN: CGCGBR. ISSN: 0301-0171.
DT Article
LA English
OS Genbank-Z46787
ED Entered STN: 20 Apr 1998
Last Updated on STN: 20 Apr 1998
AB The RING-finger is a unique zinc-chelating domain involved in mediating protein-protein interactions. The extensive sequence homology within the RING-finger domain allowed us to clone a novel member of the RING-finger family of genes. This cDNA clone, designated RNF5 (Ring-finger protein 5), contained an open reading frame of 540 nucleotides. Its predicted amino acid sequence revealed significant homology to a hypothetical protein encoded by Caenorhabditis elegans cosmid C16C10.7. The expression of RNF5 was detected in a variety of human tissues. The RNF5 gene was mapped by fluorescence in situ hybridization to chromosome 6p21.31. Radiation hybrid mapping further assigned RNF5 to a region proximal to the major histocompatibility complex (MHC) on chromosome 6. RNF5 is the third RING-finger **gene identified** in the region proximal to MHC raising the possibility that the RING-finger family of genes may exist as a cluster in this region.

L5 ANSWER 10 OF 15 MEDLINE on STN DUPLICATE 5
AN 97086640 MEDLINE
DN PubMed ID: 8932329
TI **Expression** of the **zinc finger** gene EVI-1 in ovarian and other cancers.
AU Brooks D J; Woodward S; Thompson F H; Dos Santos B; Russell M; Yang J M; Guan X Y; Trent J; Alberts D S; Taetle R
CS Department of Medicine, University of Arizona and Arizona Cancer Center, Tucson 85724, USA.
NC CA23074 (NCI)
CA41183 (NCI)
SO British journal of cancer, **(1996 Nov)** 74 (10) 1518-25.
Journal code: 0370635. ISSN: 0007-0920.
CY SCOTLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199612
ED Entered STN: 19970128
Last Updated on STN: 19990129
Entered Medline: 19961227

AB The EVI-1 **gene** was originally **detected** as an ectopic viral insertion site and encodes a nuclear zinc finger DNA-binding protein. Previous studies showed restricted EVI-1 RNA or protein expression during ontogeny; in a kidney and an endometrial carcinoma cell line; and in normal murine oocytes and kidney cells. EVI-1 expression was also detected in a subset of acute myeloid leukaemias (AMLs) and myelodysplasia. Because EVI-1 is expressed in the urogenital tract during development, we examined ovarian cancers and normal ovaries for EVI-1 RNA expression using reverse transcription polymerase chain reaction (RT-PCR) and RNAase protection. Chromosome abnormalities were examined using karyotypes and whole chromosome 3 and 3q26 fluorescence in situ hybridisation (FISH). RNA from six primary ovarian tumours, five normal ovaries and 47 tumour cell lines (25 ovarian, seven melanoma, three prostate, seven breast and one each of bladder, endometrial, lung, epidermoid and histiocytic lymphoma) was studied. Five of six primary ovarian tumours, three of five normal ovaries and 22 of 25 ovarian cell lines expressed EVI-1 RNA. A variety of other non-haematological cancers also expressed EVI-1 RNA. Immunostaining of ovarian cancer cell lines revealed nuclear EVI-1 protein. In contrast, normal ovary stained primarily within oocytes and faintly in stroma. Primary ovarian tumours showed nuclear and intense, diffuse cytoplasmic staining. Quantitation of EVI-1 RNA, performed using RNAase protection, showed ovarian carcinoma cells expressed 0 to 40 times the EVI-1 RNA in normal ovary, and 0-6 times the levels in leukaemia cell lines. Southern analyses of ovarian carcinoma cell lines showed no amplification or rearrangements involving EVI-1. In some acute leukaemias, activation of EVI-1 transcription is associated with translocations involving 3q26, the site of the EVI-1 gene. Ovarian carcinoma karyotypes showed one line with quadruplication 3(q24q27), but no other clonal structural rearrangements involving 3q26. However, whole chromsome 3 and 3q26 FISH performed on lines with high EVI-1 expression showed translocations involving chromosome 3q26. EVI-1 is overexpressed in ovarian cancer compared with normal ovaries, suggesting a role for EVI-1 in solid tumour carcinogenesis or progression. Mechanisms underlying EVI-1 overexpression remain unclear, but may include rearrangements involving chromosome 3q26.

L5 ANSWER 11 OF 15 MEDLINE on STN DUPLICATE 6
AN 96269420 MEDLINE
DN PubMed ID: 8682319
TI Identification and characterization of Zic4, a new member of the mouse Zic gene family.
AU Aruga J; Yozu A; Hayashizaki Y; Okazaki Y; Chapman V M; Mikoshiba K
CS Molecular Neurobiology Laboratory, Tsukuba Life Science Center, Institute of Physical and Chemical Research (RIKEN) Tsukuba, Ibaraki, Japan.. jaruga@rtc.riken.go.jp
SO Gene, **(1996 Jun 26)** 172 (2) 291-4.
Journal code: 7706761. ISSN: 0378-1119.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-D78174
EM 199608
ED Entered STN: 19960828
Last Updated on STN: 19960828
Entered Medline: 19960822
AB The mouse Zic genes encode **zinc-finger** (Zf) proteins **expressed** only in the cerebellum of the adult brain. The genes are the vertebrate homologues of the Drosophila pair-rule gene, odd-paired (opa). We **identified** a novel **gene**, Zic4, which belongs to the Zic gene family, through a genomic and cDNA cloning study. Zic4 is highly similar to Zic1, Zic2 and Zic3, especially in its Zf motif.

An analysis of the genomic organization of Zic4 showed that the gene shares a common exon-intron boundary with Zic1, Zic2, Zic3 and opa. The chromosomal location of Zic4 was determined to be mouse chromosome 9 in the vicinity of Zic1, using an interspecific backcross panel. An RNase protection study showed that Zic4 is expressed only in the cerebellum during the adult stage, as are the other Zic genes. The temporal profile of mRNA expression in the developing cerebellum is similar to that of Zic3 which has a peak on postnatal day 5. These findings suggest that Zic4 is a gene which works cooperatively with other Zic genes during cerebellar development.

L5 ANSWER 12 OF 15 MEDLINE on STN DUPLICATE 7
AN 96353450 MEDLINE
DN PubMed ID: 8737674
TI A new Cys2/His2 **zinc finger** gene, rKr1, **expressed** in oligodendrocytes and neurons.
AU Pott U; Colello R J; Schwab M E
CS Brain Research Institute, University of Zurich, Switzerland.. pott@codon.nih.gov
SO Brain research. Molecular brain research, **(1996 May)** 38 (1) 109-21.
Journal code: 8908640. ISSN: 0169-328X.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-U41164
EM 199702
ED Entered STN: 19970305
Last Updated on STN: 20000303
Entered Medline: 19970220
AB The myelination of nerve fibers is essential for the function of the vertebrate nervous system as a prerequisite for fast saltatory conduction of action potentials. In the central nervous system (CNS), myelin is produced by oligodendrocytes. In order to **identify gene** regulatory proteins involved in the differentiation of this glial cell type or in the expression of myelin-specific genes, we have constructed a cDNA library from a highly enriched population of rat oligodendrocytes and screened this library for members of the Kruppel family of Cys2/His2 zinc finger proteins. One of the identified clones, named rKr1, encodes a novel protein of 650 amino acids which contains 12 carboxy-terminal zinc finger domains and an amino-terminal acidic domain. On Northern blots, a single rKr1 mRNA of 4.3 kb is detected. This message is present in all adult rat tissues tested, with the highest levels found in the CNS and testis. In situ hybridization on the P15 brain revealed that the transcript is expressed in differentiated oligodendrocytes and in subtypes of neurons. Particularly high message levels are found in motor neurons of the brainstem and the spinal cord. The modular structure of the rKr1 protein, in which a potential DNA binding region (the zinc fingers) is combined with a putative activation domain (the acidic region), suggests a function as sequence-specific transcriptional activator.

L5 ANSWER 13 OF 15 MEDLINE on STN DUPLICATE 8
AN 95348803 MEDLINE
DN PubMed ID: 7623123
TI Developmental analysis of murine Promyelocyte Leukemia **Zinc Finger** (PLZF) gene **expression**: implications for the neuromeric model of the forebrain organization.
AU Avantaggiato V; Pandolfi P P; Ruthardt M; Hawe N; Acampora D; Pelicci P G; Simeone A
CS International Institute of Genetics and Biophysics, Consiglio Nazionale delle Ricerche, Naples, Italy.

SO Journal of neuroscience : official journal of the Society for Neuroscience, **(1995 Jul)** 15 (7 Pt 1) 4927-42.
Journal code: 8102140. ISSN: 0270-6474.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199508
ED Entered STN: 19950911
Last Updated on STN: 19950911
Entered Medline: 19950828
AB Promyelocyte Leukemia Zinc Finger (PLZF) is a Kruppel-like zinc finger **gene** previously **identified** in a unique case of acute promyelocytic leukemia (APL) as the counterpart of a reciprocal chromosomal translocation involving the retinoic acid receptor alpha gene (RAR alpha). PLZF is highly conserved throughout evolution from yeast to mammals. To elucidate its role, we isolated the murine PLZF gene and studied its expression during embryogenesis. PLZF is expressed in an extremely dynamic pattern with transcripts appearing at E 7.5 in the anterior neuroepithelium and quickly spreading to the entire neuroectoderm until E 10. At E 8.5, PLZF is transcribed in most of the endoderm. During mid to late gestation PLZF is expressed in restricted domains of the developing CNS as well as in specific organs and body structures. We have focused our attention on the developing forebrain where PLZF is transcribed in a transverse, segment-like domain corresponding to the anterior pretectum, in the alarmost part of the dorsal thalamus, in the epithalamus, and in the hypothalamus along a defined longitudinal subdomain. Furthermore, PLZF is expressed in several segmentary boundaries, among them, the zona limitans intrathalamica. Combined analysis with other regionally restricted genes, such as Orthopedia and Dlx1, indicates that in the hypothalamus the PLZF domain is contained within that of Orthopedia and both are complementary to that of Dlx1. Our data suggest a role for PLZF in the establishment and maintenance of transverse identities, longitudinal subdomains, and interneuromeric boundaries, providing additional evidences in favor of the neuromeric organization of the forebrain.

L5 ANSWER 14 OF 15 MEDLINE on STN DUPLICATE 9
AN 90239549 MEDLINE
DN PubMed ID: 2110381
TI **Expression** of a **zinc finger** gene in HTLV-I- and HTLV-II-transformed cells.
AU Wright J J; Gunter K C; Mitsuya H; Irving S G; Kelly K; Siebenlist U
CS Laboratory of Immunoregulation, National Institute of Allergy and Infectious Diseases, National Institutes of Health, Bethesda, MD 20892.
SO Science, **(1990 May 4)** 248 (4955) 588-91.
Journal code: 0404511. ISSN: 0036-8075.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; AIDS
OS GENBANK-M33672
EM 199006
ED Entered STN: 19900706
Last Updated on STN: 19970203
Entered Medline: 19900607
AB Gene products encoded by the human T cell leukemia virus (HTLV) types I and II mediate transformation by the transactivation of cellular genes necessary for proliferation, probably including transcriptional regulatory factors. By searching for factors that may control proliferation, a zinc finger **gene** (225) was **identified** that was constitutively expressed in all HTLV-I- or HTLV-II-transformed cell lines

examined, whereas in normal T cells it was only transiently expressed after mitogenic stimulation. The 225 gene was also constitutively expressed in two HTLV-I-transformed helper T cell clones, but not in the parental cell lines. Thus this putative cellular transcriptional factor, which was abnormally expressed in retrovirus-infected cells, may have a role in transformation.

L5 ANSWER 15 OF 15 MEDLINE on STN
AN 88196089 MEDLINE
DN PubMed ID: 3129290
TI A gene encoding a protein with zinc fingers is activated during G0/G1 transition in cultured cells.
AU Chavrier P; Zerial M; Lemaire P; Almendral J; Bravo R; Charnay P
CS Differentiation Programme, European Molecular Biology Laboratory, Heidelberg, FRG.
SO EMBO journal, **(1988 Jan)** 7 (1) 29-35.
Journal code: 8208664. ISSN: 0261-4189.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-X06746
EM 198805
ED Entered STN: 19900308
Last Updated on STN: 19970203
Entered Medline: 19880531
AB Zinc fingers are DNA-binding domains present in several eukaryotic regulatory proteins. We have **identified** a mouse **gene**, Krox-20, encoding a protein with three **zinc fingers** and whose **expression** is activated during G0/G1 transition in cultured cells. Serum stimulation of quiescent cells leads to rapid and transient accumulation of Krox-20 mRNA, with kinetics similar to those of the c-fos proto-oncogene. The induction does not require de-novo protein synthesis. In the mouse, Krox-20 is expressed at low levels in tissues which contain rapidly dividing cells. These properties suggest that Krox-20 encodes a transcription control factor, possibly involved in the modulation of cell proliferation.

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=> d his

     (FILE 'HOME' ENTERED AT 12:32:19 ON 06 MAR 2004)

     FILE 'MEDLINE, BIOSIS' ENTERED AT 12:32:29 ON 06 MAR 2004
L1           953 S ZINC FINGER AND (GENE (2A) (IDENTIF? OR DETECT?))
L2           530 S L1 AND PY<2000
L3            56 S (ZINC FINGER (2A) EXPRESS?) AND (GENE (2A) (IDENTIF? OR DETEC
L4            24 S L3 AND PY<2000
L5            15 DUPLICATE REMOVE L4 (9 DUPLICATES REMOVED)

=> s (zinc finger (2a) (regulat? or induction or induce or repress?)) and (gene
(2a) (identif? or detect?))
L6           35 (ZINC FINGER (2A) (REGULAT? OR INDUCTION OR INDUCE OR REPRESS?))
                AND (GENE (2A) (IDENTIF? OR DETECT?))

=> s l6 and py<2000
L7           16 L6 AND PY<2000

=> duplicate remove l7
DUPLICATE PREFERENCE IS 'MEDLINE, BIOSIS'
KEEP DUPLICATES FROM MORE THAN ONE FILE?  Y/(N):n
PROCESSING COMPLETED FOR L7
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L8 10 DUPLICATE REMOVE L7 (6 DUPLICATES REMOVED)

=> d 1-10 bib ab

L8 ANSWER 1 OF 10 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2000:61166 BIOSIS
DN PREV200000061166
TI Fetal Alz-50 clone 1, a novel zinc finger protein, binds a specific DNA sequence and acts as a transcriptional regulator.
AU Jordan-Sciutto, Kelly L.; Dragich, Joanna M.; Rhodes, James L.; Bowser, Robert [Reprint author]
CS Dept. of Pathology, University of Pittsburgh School of Medicine, 3500 Terrace St., BST S-420, Pittsburgh, PA, USA
SO Journal of Biological Chemistry, (Dec. 3, 1999) Vol. 274, No. 49, pp. 35262-35268. print.
CODEN: JBCHA3. ISSN: 0021-9258.
DT Article
LA English
ED Entered STN: 9 Feb 2000
Last Updated on STN: 3 Jan 2002
AB Fetal Alz-50 clone 1 (FAC1) is a novel, developmentally regulated gene that exhibits changes in protein expression and subcellular localization during neuronal development and neurodegeneration. To understand the functional implications of altered subcellular localization, we have established a normal cellular function of FAC1. The FAC1 amino acid sequence contains regional homology to transcriptional regulators. Using the polymerase chain reaction-assisted binding site selection assay, we have identified a DNA sequence recognized by recombinant FAC1. Mutation of any 2 adjacent base pairs in the identified binding site dramatically reduced the binding preference of FAC1, demonstrating that the binding is specific for the identified site. Nuclear extracts from neural and non-neural cell lines contained a DNA-binding activity with similar specificity and nucleotide requirements as the recombinant FAC1 protein. This DNA-binding activity can be attributed to FAC1 since it is dependent upon the presence of FAC1 and behaves identically on a nondenaturing polyacrylamide gel as transiently transfected FAC1. In NIH3T3 cells, luciferase reporter plasmids containing the identified binding site (CACAACAC) were repressed by cotransfected FAC1 whether the binding site was proximal or distal to the transcription initiation site. This study indicates that FAC1 is a DNA-binding protein that functions as a transcription factor when localized to the nucleus.

L8 ANSWER 2 OF 10 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 1999:449479 BIOSIS
DN PREV199900449479
TI Cloning and characterization of a novel **zinc finger** transcriptional **repressor**: A direct role of the **zinc finger** motif in **repression**.
AU He, Gong-Ping; Kim, Sungwoo; Ro, Hyo-Sung [Reprint author]
CS Department of Biochemistry, Faculty of Medicine, Dalhousie University, Sir Charles Tupper Medical Building, Halifax, NS, B3H 4H7, Canada
SO Journal of Biological Chemistry, (May 21, 1999) Vol. 274, No. 21, pp. 14678-14684. print.
CODEN: JBCHA3. ISSN: 0021-9258.
DT Article
LA English
OS Genbank-AF090326
ED Entered STN: 26 Oct 1999
Last Updated on STN: 3 May 2000
AB We have identified a novel transcriptional repressor, AEBP2, that binds to a regulatory sequence (termed AE-1) located in the proximal promoter region of the aP2 gene that encodes the adipose fatty acid-binding

protein. Sequence analysis of AEBP2 cDNA revealed that it encodes a protein containing three Gli-Kruppel (Cys2-His2)-type zinc fingers. Northern blot analysis revealed two transcripts (4.5 and 3.5 kilobases) which were ubiquitously expressed in every mouse tissue examined. In co-transfection assays, AEBP2 repressed transcription from the homologous aP2 promoter containing multiple copies of the AE-1 sequence. Moreover, a chimeric construct encoding a fusion AEBP2 protein with the Gal4 DNA-binding domain was able to repress the transcriptional activity of a heterologous promoter containing the Gal4-binding sequence. The transcriptional repression function of AEBP2 was completely abolished when one of the conserved histidine residues and a flanking serine residue in the middle zinc finger were replaced with an arginine residue. The defective transcriptional repression function of the mutant derivative was due neither to lack of expression nor to a failure to localize to the nucleus. Moreover, both the wild-type and mutant derivative of either the histidine-tagged recombinant AEBP2 proteins or the in vitro translated Gal4-AEBP2 fusion proteins were equally able to bind to the target DNA. These results suggest that a portion of the zinc finger structure may play a direct role in transcriptional repression function, but not in DNA binding.

L8 ANSWER 3 OF 10 MEDLINE on STN DUPLICATE 1
AN 1999160897 MEDLINE
DN PubMed ID: 10049742
TI Analysis of the consensus binding sequence and the DNA-binding domain of ZF5.
AU Obata T; Yanagidani A; Yokoro K; Numoto M; Yamamoto S
CS Department of Dermatology, Hiroshima University School of Medicine, Japan.. tobata@ipc.hiroshima-u.ac.jp
SO Biochemical and biophysical research communications, **(1999 Feb 16)** 255 (2) 528-34.
Journal code: 0372516. ISSN: 0006-291X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199903
ED Entered STN: 19990324
Last Updated on STN: 19990324
Entered Medline: 19990311
AB Murine ZF5 is a transcription factor with five **zinc finger** motifs that **represses** the c-myc gene by binding to two GC-rich elements at the promoter region. Because of its ubiquitous expression in a variety of tissues, elucidation of biological functions and cellular target genes of ZF5 is of great interest. As the first step of **identifying** cellular target **genes**, we have attempted to determine the consensus binding motif for ZF5. We succeeded in isolating 19 oligonucleotide duplex DNAs to which ZF5 binds and determined the binding sequences with DNase I footprinting analysis. From these sequences, we deduced the consensus binding motif for ZF5 to be GSGCGCGR. In addition, we have analyzed the DNA-binding domain of ZF5 by testing a series of deletion mutants. It turned out that the zinc fingers 3 and 4 of the five finger motifs play a critical role in DNA binding. Copyright 1999 Academic Press.

L8 ANSWER 4 OF 10 MEDLINE on STN DUPLICATE 2
AN 1999389731 MEDLINE
DN PubMed ID: 10458916
TI Cloning and characterization of ZNF236, a glucose-**regulated** Kruppel-like **zinc-finger** gene mapping to human chromosome 18q22-q23.
AU Holmes D I; Wahab N A; Mason R M

CS Division of Biomedical Sciences, Imperial College School of Medicine, BMS Building, South Kensington, London, SW7 2AZ, United Kingdom.
SO Genomics, **(1999 Aug 15)** 60 (1) 105-9.
Journal code: 8800135. ISSN: 0888-7543.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-AF085243; GENBANK-AF085244
EM 199909
ED Entered STN: 19991012
Last Updated on STN: 19991012
Entered Medline: 19990930
AB We report the cDNA cloning and characterization of ZNF236, a novel Kruppel-like zinc-finger **gene** initially **identified** by its glucose-regulated expression in human mesangial cells using mRNA differential display. Using the differential display fragment as a probe, we screened a human fetal kidney cDNA library and isolated several clones representing two differently spliced mRNA transcripts, designated ZNF236a and -b. Both transcripts were identical apart from the presence of an additional exon in ZNF236a that truncates the open reading frame. RT-PCR analysis confirmed the expression of both transcripts to be upregulated in human mesangial cells in response to elevated levels of d-glucose. ZNF236a and -b cDNAs encode polypeptides of 174 and 204 kDa, containing 25 and 30 C(2)H(2) zinc-finger motifs, respectively. Northern blot analysis showed that ZNF236 is ubiquitously expressed in all human tissues tested. Expression levels were highest in skeletal muscle and brain, intermediate in heart, pancreas, and placenta, and lowest in kidney, liver, and lung. Southern zoo blot analysis indicated that ZNF236 is conserved in the genomes of all mammalian species tested, but not in yeast. The mapping of ZNF236 to human chromosome 18q22-q23, close to the IDDM6 locus, coupled with the glucose-regulated expression of the gene in human mesangial cells, suggests that ZNF236 may be a candidate gene for diabetic nephropathy.

L8 ANSWER 5 OF 10 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 1998:490677 BIOSIS
DN PREV199800490677
TI The early growth response protein (EGR-1) regulates interleukin-2 transcription by synergistic interaction with the nuclear factor of activated T cells.
AU Decker, Eva L.; Skerka, Christine; Zipfel, Peter F. [Reprint author]
CS Research Group Biomolecular Med., Bernhard Nocht Inst. Tropical Med., Bernhard-Nocht-Strasse 74, D-20359 Hamburg, Germany
SO Journal of Biological Chemistry, (Oct. 9, 1998) Vol. 273, No. 41, pp. 26923-26930. print.
CODEN: JBCHA3. ISSN: 0021-9258.
DT Article
LA English
ED Entered STN: 18 Nov 1998
Last Updated on STN: 18 Nov 1998
AB The early growth response-1 gene (EGR-1) is induced by a wide range of stimuli in diverse cell types; however, EGR-1-regulated genes display a highly restricted pattern of expression. Recently, an overlapping Sp1-EGR-1 binding site has been identified within the interleukin-2 (IL-2) gene promoter directly upstream of the binding site for the nuclear factor of activated T cells (NFAT). We used transfection assays to study how the abundantly and constitutively expressed Sp1 protein and the immediate early EGR-1 **zinc finger** protein **regulate** IL-2 gene expression. Here, we identify EGR-1 as an important activator of the IL-2 gene. In Jurkat T cells, EGR-1 but not Sp1 acts as a potent

coactivator for IL-2 transcription, and in combination with NFATc, EGR-1 increases transcription of an IL-2 reporter construct 200-fold. Electrophoretic mobility shift assays reveal that recombinant EGR-1 and NFATc bind independently to their target sites within the IL-2 promoter, and the presence of both sites on the same DNA molecule is required for EGR-1cntdotNFATccntdotDNA complex formation. The transcriptional synergy observed here for EGR-1 and NFATc explains how the abundant nuclear factor EGR-1 contributes to the expression of restrictively expressed genes.

L8 ANSWER 6 OF 10 MEDLINE on STN DUPLICATE 3
AN 1998381937 MEDLINE
DN PubMed ID: 9717837
TI Common mutations in autoimmune polyendocrinopathy-candidiasis-ectodermal dystrophy patients of different origins.
AU Scott H S; Heino M; Peterson P; Mittaz L; Lalioti M D; Betterle C; Cohen A; Seri M; Lerone M; Romeo G; Collin P; Salo M; Metcalfe R; Weetman A; Papasavvas M P; Rossier C; Nagamine K; Kudoh J; Shimizu N; Krohn K J; Antonarakis S E
CS Department of Genetics and Microbiology, University of Geneva Medical School, Switzerland.. Hamish.Scott@medecine.unige.ch
SO Molecular endocrinology (Baltimore, Md.), **(1998 Aug)** 12 (8) 1112-9.
Journal code: 8801431. ISSN: 0888-8809.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199810
ED Entered STN: 19990106
Last Updated on STN: 19990106
Entered Medline: 19981029
AB Autoimmune polyendocrinopathy-candidiasis-ectodermal dystrophy (APECED; OMIM *240300, also called APS 1,) is a rare autosomal recessive disorder that is more frequent in certain isolated populations. It is generally characterized by two of the three major clinical symptoms that may be present, Addison's disease and/or hypoparathyroidism and/or chronic mucocutaneous candidiasis. Patients may also have a number of other clinical symptoms including chronic gastritis, gonadal failure, and rarely, autoimmune thyroid disease and insulin-dependent diabetes mellitus. We and others have recently **identified** the **gene** for APECED, which we termed AIRE (for autoimmune regulator). AIRE is expressed in thymus, lymph nodes, and fetal liver and encodes a protein containing motifs suggestive of a transcriptional **regulator**, including two **zinc finger** motifs (PHD finger), a proline-rich region, and three LXXLL motifs. Six mutations, in cluding R257X, the predominant Finnish APECED allele, have been defined. R257X was also observed in non-Finnish APECED patients occurring on different chromosomal haplotypes suggesting different mutational origins. Here we present mutation analyses in an extended series of patients, mainly of Northern Italian origin. We have detected 12 polymorphisms, including one amino acid substitution, and two additional mutations, R203X and X546C, in addition to the previously described mutations, R257X, 1096-1097insCCTG, and a 13-bp deletion (1094-1106del). R257X was also the common mutation in the Northern Italian patients (10 of 18 alleles), and 1094-1106del accounted for 5 of 18 Northern Italian alleles. Both R257X and 1094-1106del were both observed in patients of four different geo-ethnic origins, and both were associated with multiple different haplotypes using closely flanking polymorphic markers showing likely multiple mutation events (six and four, respectively). The identification of common AIRE mutations in different APECED patient groups will facilitate its genetic diagnosis. In addition, the polymorphisms presented provide the tools for investigation of the

involvement of AIRE in other autoimmune diseases, particularly those affecting the endocrine system.

L8 ANSWER 7 OF 10 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 1998:174527 BIOSIS
DN PREV199800174527
TI Salt tolerance in crop plants: New approaches through tissue culture and gene regulation.
AU Winicov, Ilga [Reprint author]; Bastola, Dhundy R.
CS Dep. Microbiology, Univ. Nevada Reno, Reno, NV 89557, USA
SO Acta Physiologiae Plantarum, (1997) Vol. 19, No. 4, pp. 435-449. print.
CODEN: APPLDE. ISSN: 0137-5881.
DT Article
General Review; (Literature Review)
LA English
ED Entered STN: 20 Apr 1998
Last Updated on STN: 20 Apr 1998
AB Recent approaches to study of salinity tolerance in crop plants have ranged from genetic mapping to molecular characterization of gene products induced by salt/drought stress. Transgenic plant design has allowed to test the effects of overexpression of specific prokaryotic or plant genes that are known to be up-regulated by salt/drought stress. This review summarizes current progress in the field in the context of adaptive metabolic and physiological responses to salt stress and their potential role in long term tolerance. Specifically considered are gene activation by salt, in view of proposed avenues for improved salt tolerance and the need to ascertain the additional influences of developmental regulation of such genes. Discussion includes the alternate genetic strategy we have pursued for improving salinity tolerance in alfalfa (Medicago saliva L.) and rice (Oryza sativa L.). This strategy combines single-step selection of salt-tolerant cells in culture, followed by regeneration of salt-tolerant plants and **identification** of **genes** important in conferring salt tolerance. We have postulated that activation or improved expression of a subset of genes encoding functions that are particularly vulnerable under conditions of salt-stress could counteract the molecular effects of such stress and could provide incremental improvements in tolerance. We have proceeded to identify the acquired specific changes in gene regulation for our salt-tolerant mutant cells and plants. One particularly interesting and novel gene isolate from the salt-tolerant cells is Alfin1, which encodes a putative **zinc-finger regulatory** protein, expressed predominantly in roots. We have demonstrated that this protein binds DNA in a sequence specific manner and may be potentially important in gene regulation in roots in response to salt and an important marker for salt tolerance in crop plants.

L8 ANSWER 8 OF 10 MEDLINE on STN DUPLICATE 4
AN 97098643 MEDLINE
DN PubMed ID: 8943320
TI Cleavage of RNA hairpins mediated by a developmentally **regulated** CCCH **zinc finger** protein.
AU Bai C; Tolias P P
CS Public Health Research Institute, New York, New York 10016, USA.
SO Molecular and cellular biology, **(1996 Dec)** 16 (12) 6661-7.
Journal code: 8109087. ISSN: 0270-7306.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-AF033201
EM 199701
ED Entered STN: 19970219

Last Updated on STN: 20000303
Entered Medline: 19970131

AB Control of RNA turnover is a major, but poorly understood, aspect of gene regulation. In multicellular organisms, progress toward dissecting RNA turnover pathways has been made by defining some cis-acting sequences that function as either regulatory or cleavage targets (J. G. Belasco and G. Brawerman, Control of Messenger RNA Stability, 1993). However, the **identification** of **genes** encoding proteins that regulate or cleave target RNAs has been elusive (C. A. Beelman and R. Parker, Cell 81:79-183, 1995); this gap in knowledge has made it difficult to identify additional components of RNA turnover pathways. We have utilized a modified expression cloning strategy to identify a developmentally regulated gene from Drosophila melanogaster that encodes a RNase that we refer to as Clipper (CLP). Significant sequence matches to open reading frames encoding unknown functions identified from the Caenorhabditis elegans and Saccharomyces cerevisiae genome sequencing projects suggest that all three proteins are members of a new protein family conserved from lower eukaryotes to invertebrates. We demonstrate that a member of this new protein family specifically cleaves RNA hairpins and that this activity resides in a region containing five copies of a previously uncharacterized CCCH zinc finger motif. CLP's endoribonucleolytic activity is distinct from that associated with RNase A (P. Blackburn and S. Moore, p. 317-433, in P. D. Boyer, ed., The Enzymes, vol. XV, part B, 1982) and is unrelated to RNase III processing of rRNAs and tRNAs (J. G. Belasco and G. Brawerman, Control of Messenger RNA Stability, 1993, and S. A. Elela, H. Igel, and M. Ares, Cell 85:115-124, 1995). Our results suggest that CLP may function directly in RNA metabolism.

L8 ANSWER 9 OF 10 MEDLINE on STN DUPLICATE 5
AN 96026007 MEDLINE
DN PubMed ID: 7565779
TI Role of EGR-1 in thapsigargin-inducible apoptosis in the melanoma cell line A375-C6.
AU Muthukkumar S; Nair P; Sells S F; Maddiwar N G; Jacob R J; Rangnekar V M
CS Department of Surgery, University of Kentucky, Lexington 40536, USA.
NC CA52837 (NCI)
SO Molecular and cellular biology, **(1995 Nov)** 15 (11) 6262-72.
Journal code: 8109087. ISSN: 0270-7306.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199511
ED Entered STN: 19951227
Last Updated on STN: 20000303
Entered Medline: 19951121

AB Induction of apoptosis by diverse exogenous signals is dependent on elevation of intracellular Ca2+. This process of cell death can be blocked by actinomycin D, indicating that it requires gene transcription events. To **identify genes** that are required for apoptosis, we used thapsigargin (TG), which inhibits endoplasmic reticulum-dependent Ca(2+)-ATPase and thereby increases cytosolic Ca2+. Exposure to TG led to **induction** of the **zinc finger** transcription factor, EGR-1, and apoptosis in human melanoma cells, A375-C6. To determine the functional relevance of EGR-1 expression in TG-inducible apoptosis, we employed a dominant negative mutant which functionally competes with EGR-1 in these cells. Interestingly, the dominant negative mutant inhibited TG-inducible apoptosis. Consistent with this observation, an antisense oligomer directed against Egr-1 also led to a diminution of the number of cells that undergo TG-inducible apoptosis. These results suggest a novel regulatory role for EGR-1 in mediating apoptosis that is induced by

intracellular Ca2+ elevation. We have previously shown that in these melanoma cells, EGR-1 acts to inhibit the growth arresting action of interleukin-1. Together, these results imply that EGR-1 plays inducer-specific roles in growth control.

L8 ANSWER 10 OF 10 MEDLINE on STN DUPLICATE 6
AN 94165093 MEDLINE
DN PubMed ID: 8120052
TI A novel nuclear protein with **zinc fingers** down-**regulated** during early mammalian cell differentiation.
AU Okazaki S; Tanase S; Choudhury B K; Setoyama K; Miura R; Ogawa M; Setoyama C
CS Department of Biochemistry, Kumamoto University School of Medicine, Japan.
SO Journal of biological chemistry, **(1994 Mar 4)** 269 (9) 6900-7.
Journal code: 2985121R. ISSN: 0021-9258.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-D21850
EM 199404
ED Entered STN: 19940412
Last Updated on STN: 19980206
Entered Medline: 19940404
AB We introduced a promoter trap vector carrying a neo gene as a selectable marker into F9 cells and established several cell lines in which the expression of neo gene is under the control of an endogenous host gene that is active only in the undifferentiated F9 cells. Using one of these cell lines, G19, we isolated the integrated neo construct and its flanking host sequences by the plasmid rescue method, **identified** the host **gene** which contributes to the expression of neo gene, and named it the Zfp-57 gene. Two different Zfp-57 transcripts (1.8 and 3.2 kilobases) were identified in the undifferentiated F9 cells, and the levels of these transcripts were decreased significantly within a short time after induction of differentiation. We examined mouse organs for the presence of the Zfp-57 RNAs and found that the 1.8-kilobase RNA was detected only in the testis. The Zfp-57 cDNAs corresponding to the two different RNAs were isolated, and a comparison of the nucleotide sequences revealed that their coding regions were completely identical, but they differed both in length and in sequence of the 3'-untranslated region. The Zfp-57 cDNA encoded a protein consisting of 421 amino acids with an extremely high content of basic amino acid residues and multiple zinc finger motifs. Immunocytochemical analysis revealed that this protein is localized in the nucleus. These findings suggest that the Zfp-57 protein is a DNA-binding protein.

=> FIL STNGUIDE

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	45.62	45.83

FILE 'STNGUIDE' ENTERED AT 12:52:42 ON 06 MAR 2004

FILE CONTAINS CURRENT INFORMATION.
LAST RELOADED: Feb 27, 2004 (20040227/UP).

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COST IN U.S. DOLLARS	SINCE FILE	TOTAL

	ENTRY	SESSION
FULL ESTIMATED COST	0.06	45.89

FILE 'MEDLINE' ENTERED AT 12:52:48 ON 06 MAR 2004

FILE 'BIOSIS' ENTERED AT 12:52:48 ON 06 MAR 2004

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=> LOG Y

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	1.23	47.12

STN INTERNATIONAL LOGOFF AT 12:52:58 ON 06 MAR 2004